Panbela™ THERAPEUTICS + CANCER PREVENTION PHARMACEUTICALS

Combined Company Investor Presentation

January 2023

Last modified on
1/18/23

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This presentation includes information about investigational agents. The efficacy and safety of such investigational agents have not yet been established. Drug development is uncertain and investigative agents may be terminated along the development process.

Combined Company Presents A Unique Opportunity: Multi-Targeted Approach to Reset Dysregulated Biology

Scientific focus on polyamines, key regulators of normal biology altered in many disease states

1. Multiple near term inflection points

2. Late-stage programs are orphan oncology related: pancreatic cancer and FAP (familial adenomatous polyposis)
   - Combined pipeline spans from pre-clinical to Phase III registration programs

3. Approximately a $5 Billion aggregate market potential across lead indications

4. Commercial partner for Phase III FAP program: fully funded registration program in the US
   - Company retains ROW rights

5. Strategic synergies and partnerships (NCI, SWOG, COG, JDRF)

Combined clinical program pipeline to create significant shareholder value

NCI-National Cancer Institute; SWOG-Southwest Oncology Group; COG-Children's Oncology Group; JDRF – Juvenile Diabetes Research Foundation

Highly Experienced Management Team with Proven Track Record

Proven orphan and oncology drug discovery, development and commercialization expertise

Dr. Jennifer Simpson
Chief Executive Officer

15+ Years

Sue Horvath
Chief Financial Officer

15+ Years

Rachel Bragg
VP, Clinical Development

14+ Years

Dr. Elizabeth Bruckheimer
VP, Chief Scientific Officer

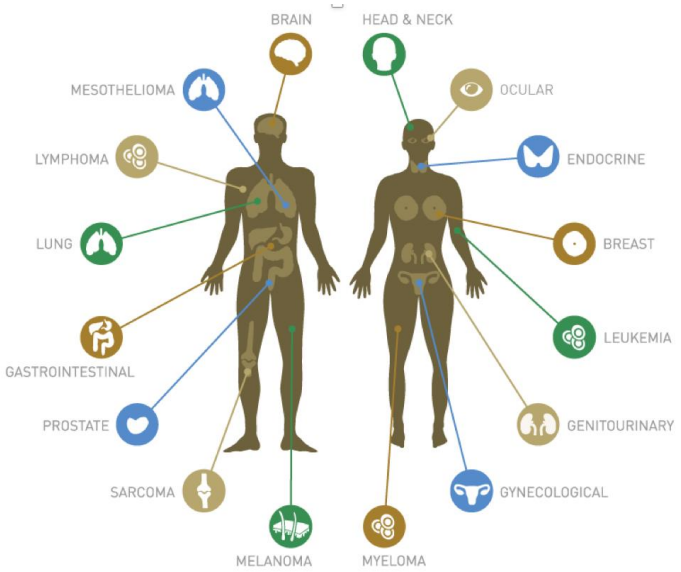
13+ Years

Dr. Ashok Chavan
VP, CMC, QA, Supply Chain

15+ Years

CombinedCo Team collectively has 10+ FDA approvals and decades of Pharma experience positioning Panbela to execute on the clinical development programs

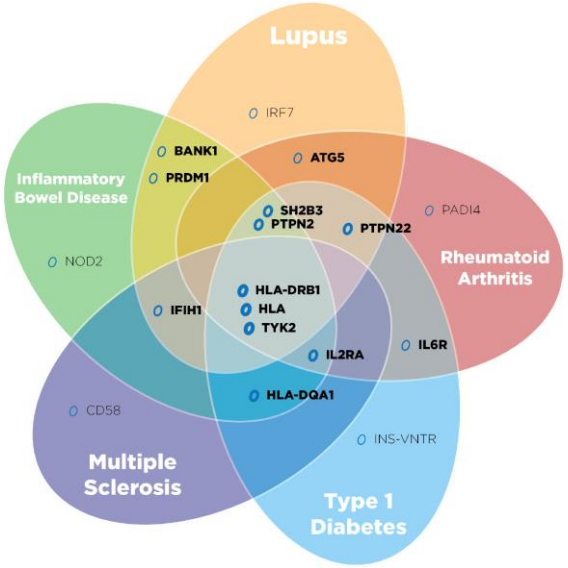
Dysregulation of the Polyamine Pathway Leads to Disease



Cancer

Pancreatic
Familial Adenomatous Polyposis (FAP)
Colorectal
Ovarian
STK11 Mutant NSCLC
Neuroblastoma

Polyamine Dysregulation in Disease



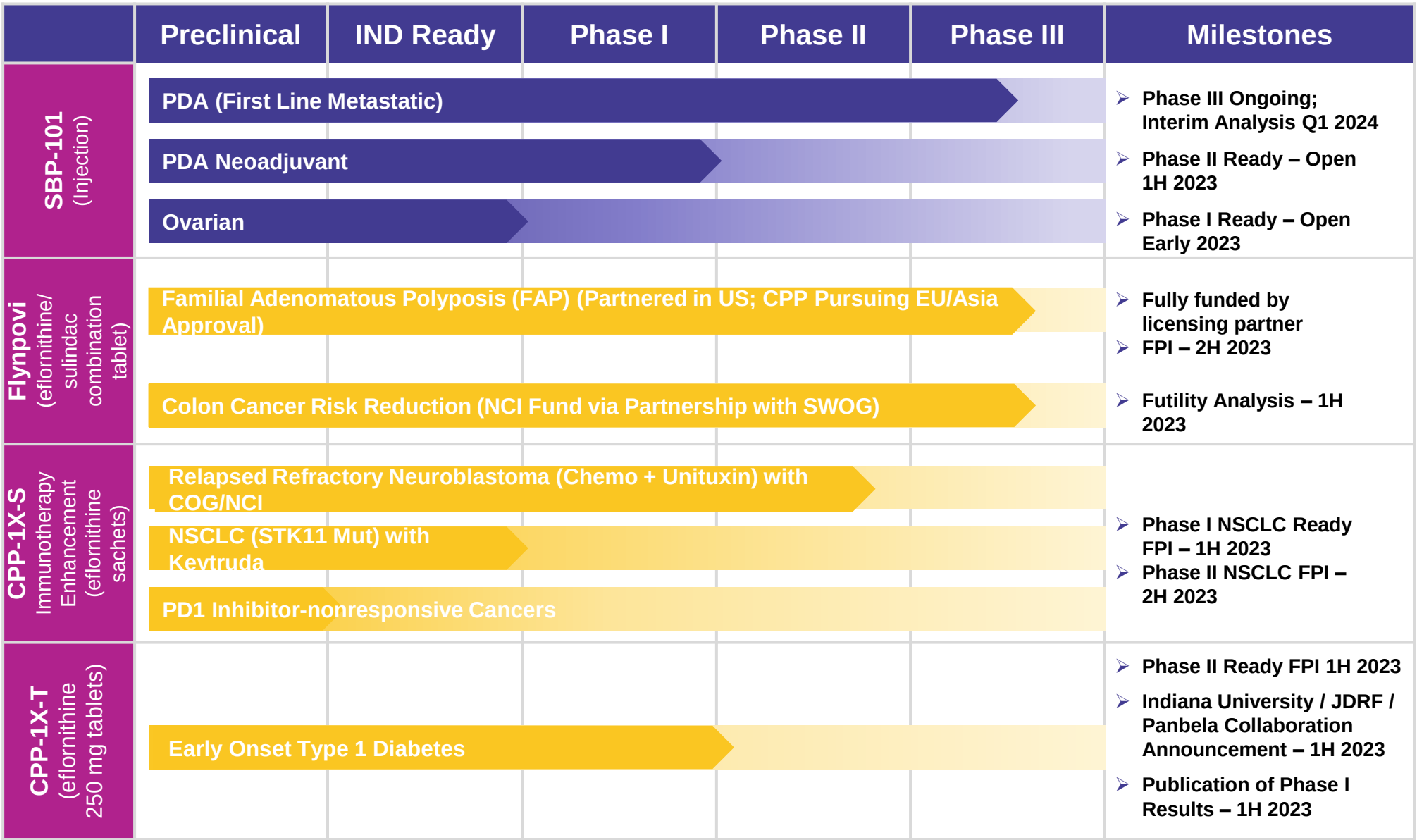
Autoimmune Diseases

Recent Onset Type 1 Diabetes

Initial focus on oncology and autoimmune disease - enhancing anti-tumor activity, preventing tumor growth, and modulating the immune system

http://ibeliveit.ca/Personalized-Cancer-Medicine/Funding-byCancer-Typr.aspx
https://www.benaroyaresearch.org/blog/post/mystery-multiple-autoimmune-diseases
NSCLC- Non Small Cell Lung Cancer

Combined Pipeline May Address Unmet Needs



PDA – Pancreatic Ductal Adenocarcinoma; FPI-First Patient In

Potential Market Opportunity for Lead Programs

Pancreatic Cancer	FAP	Colorectal Cancer	Ovarian Cancer
Incidence of 1L Metastatic Pancreatic Cancer **57K in US** **~190K in EU**	**Incidence of FAP** **~30K in US** **~50K in EU**	**Incidence of Colorectal Cancer** **~150K in US** **~340K in EU**	**Incidence of Ovarian Cancer** **~22K in US** **~79K in EU**
~247K	**~80K**	**~490K**	**~101K**
1L Metastatic Pancreatic Cancer Target Population **~32K in US** **~133K in EU**	**FAP Prevalence(1) and Target Population** **~15K in US** **~20K in EU**	**5-year prevalence(2) Colorectal Cancer Target Population** **~400K in US** **~950K in EU**	**Ovarian Cancer Target Population** **~12K in US** **~46K in EU**
~165K	**~35K**	**~1.35M**	**~58K**
Approximately 70% patients are treated.			**Approximately 58% patients are late stage at diagnosis**

FAP Source: Burt et al 2010; Varesco et al 2004; multiple KOL sources.
Pancreatic Cancer Source: American Cancer Society. Cancer Facts & Figures 2021. Atlanta, GA: American Cancer Society; 2021 and European Cancer Information Systems data 2020 (https://ecis.jrc.ec.europa.eu/).
Colorectal Cancer Source: NCI Seer statistics 2018 and https://ecis.jrc.ec.europa.eu., https://seer.cancer.gov/csr/1975_2018/browse_csr.php?sectionSEL=1&pageSEL=sect_01_table.21 and GLOBOCAN 2020.
Ovarian Cancer Source: American Cancer Society. Cancer Facts & Figures 2021. Atlanta, GA: American Cancer Society; 2021 and European Cancer Information Systems data 2020 (https://ecis.jrc.ec.europa.eu/).
1) Based on 1/10,000 prevalence, excludes 5% with no APC mut. Includes ages 15-74 and only patients with intact colon or retained rectum (~70% of FAP patients).
2) Prevalence is for "1st invasive tumor ever".

Complementary Pharmacotherapies Targeting Dysregulation

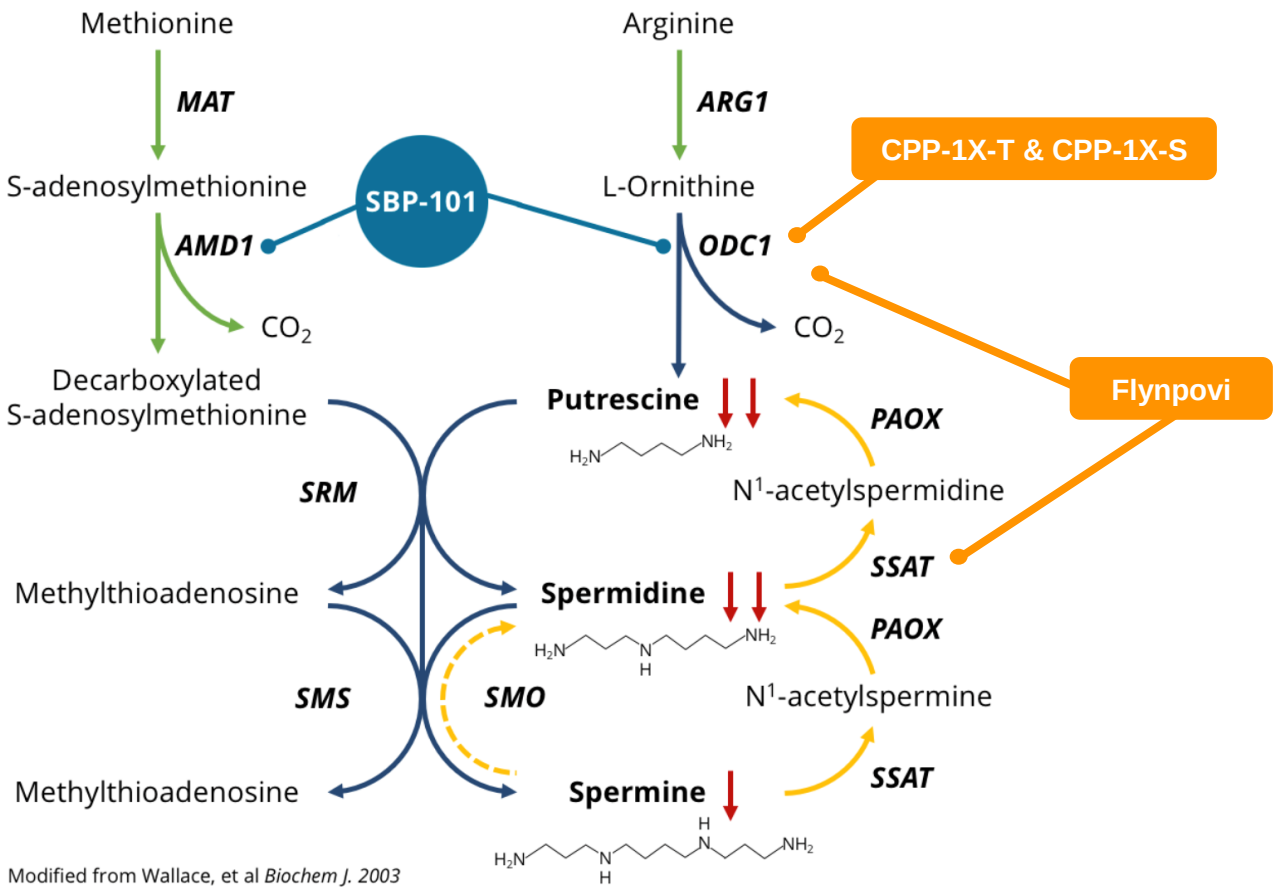
Pipeline Objective

The objective of Panbela's pipeline is the utilization of pharmacotherapies to reduce/normalize increased disease-associated polyamines using complementary pharmacotherapies

Current Pipeline

1. SBP-101
2. Flynpovi
3. CPP-1X-T
4. CPP-1X-S

Combined pipeline pharmacotherapies hit different targets in the polyamine pathway



AMD1 = Adenosylmethionine decarboxylase 1; ARG1 = Arginase 1; MAT = Methionine adenosyltransferase; ODC1 = Ornithine decarboxylase 1; PAOX = Polyamine oxidase; SMO = Spermine oxidase; SMS = Spermine synthase; SRM = Spermidine synthase; SSAT = Spermidine/spermine N^1-acetyltransferase.

SBP-101

Preliminary Efficacy of SBP-101 + Gemcitabine/Nab-paclitaxel in First-Line Metastatic Pancreatic Ductal Adenocarcinoma

	BEST OVERALL RESPONSE				Overall Response	Disease Control
	CR	PR	SD	PD		
SBP-101 (0.40 mg/kg) + G/A* (Ph Ia COHORT 2) n=7	0	5 (71%)	2 (29%)	0	**5/7 (71%)**	**5/7 (71%)**
SBP-101 (0.40 mg/kg) + G/A* (Ph Ia COHORT 4 + Ph 1b) n=29	1 (3%)	13 (45%)	10 (34%)	5 (17%)	**14/29 (48%)**	**24/29 (83%)**
Gemcitabine + Nab-paclitaxel (G/A*) n=431**	<1%	23%	27%	20%	**23%**	**48%**

	PFS			OS		
SBP-101 (0.40 mg/kg) + G/A Cohort	Ph1a 2	**Ph Ia 4+Ph Ib**	G+A*	Ph Ia 2	**Ph Ia 4+Ph Ib**	G+A*
Median (mo)	5.6	**6.5**	5.5	10.3	**14.6*** **	8.5
6 mo (%)	43	**54**	44	100%	**86%**	67%
12 mo (%)	0	**18**	16	43%	**55%**	35%

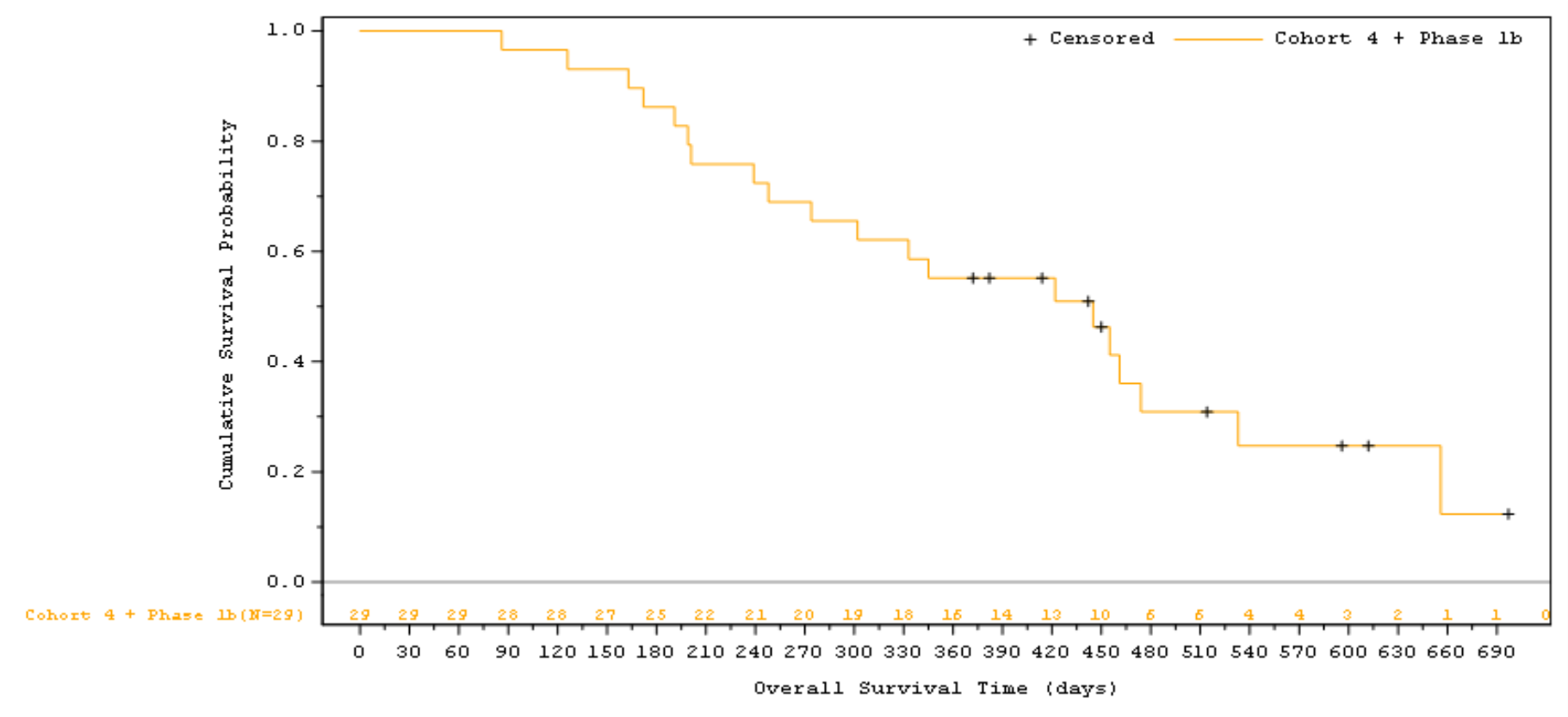
*G/A = gemcitabine + Nab-paclitaxel
**Historical control data, MPACT study G+A arm, N=431 - Source: Von Hoff NEJM 2013
*** Final Data–3/18/22
PhIa = Phase Ia
PhIb = Phase Ib
CR-Complete Response; PR-Partial Response; SD-Stable Disease
Disease Control Rate = CR+PR+SD for > 16 weeks

Efficacy Comparison

Treatment	OS	PFS	ORR	DCR	PD
CL-SBP-101-03[1]	14.6 months	6.5 months	48.3%	82.8%	4.8%
FOLFIRINOX[2]	11.1 months	6.4 months	31.6%	70.2%	15.2%
Modified FOLFIRINOX[3]	10.2 months	6.1 months	35.1%	86.5%	13.5%
Gemcitabine + Abraxane – Phase 1/2[4]	12.2 months	7.9 months	48%	68%	16%
Gemcitabine + Abraxane (MPACT)[5]	8.5 months	5.5 months	23%	48%	20%

1. CL-SBP-101-03 CSR (modified Efficacy Evaluable data n=29);
2. Conroy et al, 2011;
3. Stein et al, 2016 (MPC data);
4. Von Hoff et al, 2011;
5. Von Hoff et al, 2013

SBP-101 + Gemcitabine/Nab-paclitaxel Overall Survival*



Cohort 2 N=7: 2 patients with long term survival.

- **One still alive at 33.1 months**
- **One deceased at 30.3 months**

Cohort 4+1b = 6 patients still alive

- **One complete response (Recist)**
- **One clinical complete response (no detectable tumor)**

* Data as of 03/18/2022

SBP-101 + Gemcitabine/Nab-paclitaxel Demonstrated a Similar Safety Profile to Gemcitabine/Nab-paclitaxel in First-Line Metastatic Pancreatic Ductal Adenocarcinoma

Safety Results

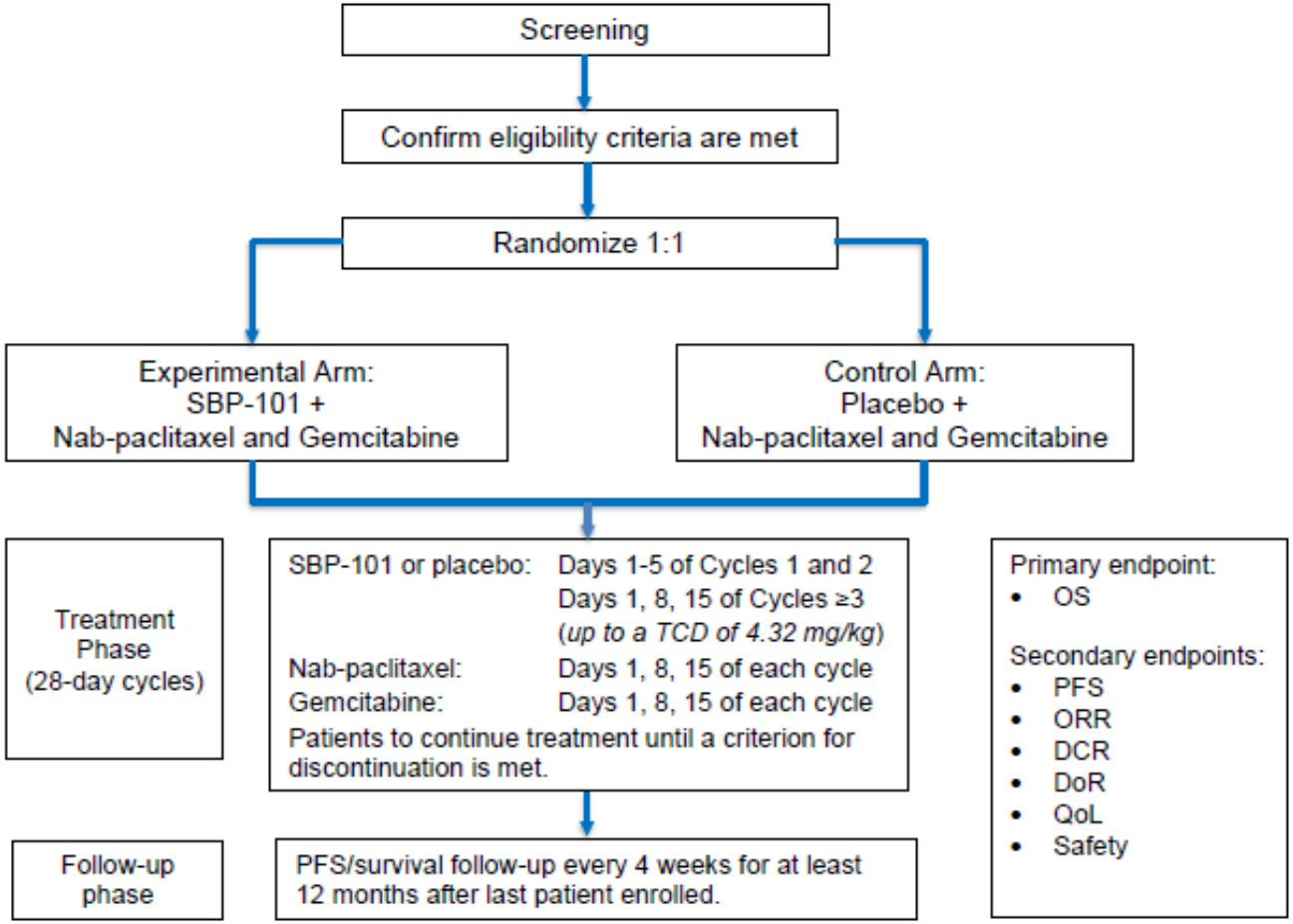
Grade ≥3 AEs of Special Interest	N	SBP-101%		G+A %**
Hematologic Events				
Neutropenia	20	40%		38%
Leukopenia	0	-		31%
Anemia	9	18%		13%
Thrombocytopenia	1	2%		17%
Febrile Neutropenia	1	2%		3%
Non-hematologic Events				
Diarrhea	7	14%		6%
Fatigue	6	12%		17%
Peripheral Neuropathy	3	6%		17%

Grade ≥3 adverse events attributable to any study medication, N=50.				
Event	**SBP-101**	**G+A****	**All 3**	**Total N (%)**
Neutropenia	0	19 (1G, 18 G+A)	1	20 (40%)
Elevated LFTs	5	0	9	14 (28%)
Anemia	0	7 (G+A)	0	9 (18%)
Diarrhea	0	6 (4A, 2G+A)	1	7 (14%)
Fatigue	0	4 (G+A)	2	6 (12%)
Vision events	4	1 (G)	2	7(14%)
Dehydration	2	2 (1A, 1 G+A)	0	4 (8%)
Peripheral neuropathy	0	3 (A)	0	3 (6%)

*Historical control data, MPACT study, G+A arm, N= 431 - Source: Von Hoff 2013
** G/A = gemcitabine + Nab-paclitaxel

Phase III Pancreatic Cancer Trial in Subjects Previously Untreated for Metastatic Pancreatic Ductal Adenocarcinoma

A Randomized, Double-Blind, Placebo Controlled Study of Nab-Paclitaxel and Gemcitabine with or Without SBP-101



OS-Overall Survival
PFS-Progression Free Survival
ORR – Overall Response Rate
DCR-Disease Control Rate
DoR – Duration of Response
QoL-Quality of Life

FLYNPOVI: A combination of CPP-1X and Sulindac

Lead Product Opportunity – CPP-1X and Sulindac (Flynpovi)

Familial Adenomatous Polyposis (FAP)

- A genetic disease caused by APC mutations where colon polyps develop early in life
- Nearly 100% of patients will develop colon cancer if the polyps are left untreated

Prevalence

1-in-10,000

~30K in US

~50K in EU

Global Markets Opportunity

Note: APC = Adenomatous polyposis coli.

Key Information:

- No approved FAP drugs on the market
- Target Physicians: Gastroenterologists
- US-only potential revenue at market share of 35%-60% (approximately 11,300-18,400 potential patients)

Pricing:

Annual Price	US only Potential Annual Revenue
$20,000	$227M - $368M
$25,000	$284M - $460M
$30,000	$341M - $552M
$35,000	$397M - $644M

North American Royalties from Licensee:

- Capped: 25% up to $100 million in total payments
- Uncapped: 5% after capped royalty is reached

Market:

- FAP affects all ethnicities
- In addition to the US - Significant market opportunity in Europe, China and Japan

FAP-310 Trial: “Time to Delay FAP-related Event” Endpoint

CPP FAP 310 global study

150 Patients Required
(171 Patients Enrolled)
1:1:1 Randomization
Two Years of Daily Treatment

CPP-1X (750 mg) + Sulindac (150 mg)	CPP-1X (750 mg) + Placebo	Placebo + Sulindac (150 mg)

Endpoint “Time to FAP-related Event” (e.g., Colectomy, Duodenectomy, Spigelman score*, etc.) over at least 24 months at any Disease Site per Patient

- Time to delay FAP-related events
  Composite Endpoint including surgeries, polyp removal, and upper GI scoring system (“Spigelman”)*; based on FDA/EMA recommendation-first ever “event” trial in FAP
- Surgical endpoints most meaningful
- *p* value with all elements of composite endpoint = 0.28 (i.e., with unvalidated non-surgery scoring system* included)
- ***p* = <0.02** in delaying surgical and interventional events in “lower GI”

* Determined to not be a “clinically meaningful” metric resulting in a change in the clinical management or treatment for the patient

Burke et al 2021 NEJM
Balaguer et al 2021 DCR

No Lower GI Surgeries for Flynpovi Combination

Event Rate Distribution of FAP-Related Surgery Events in Lower GI

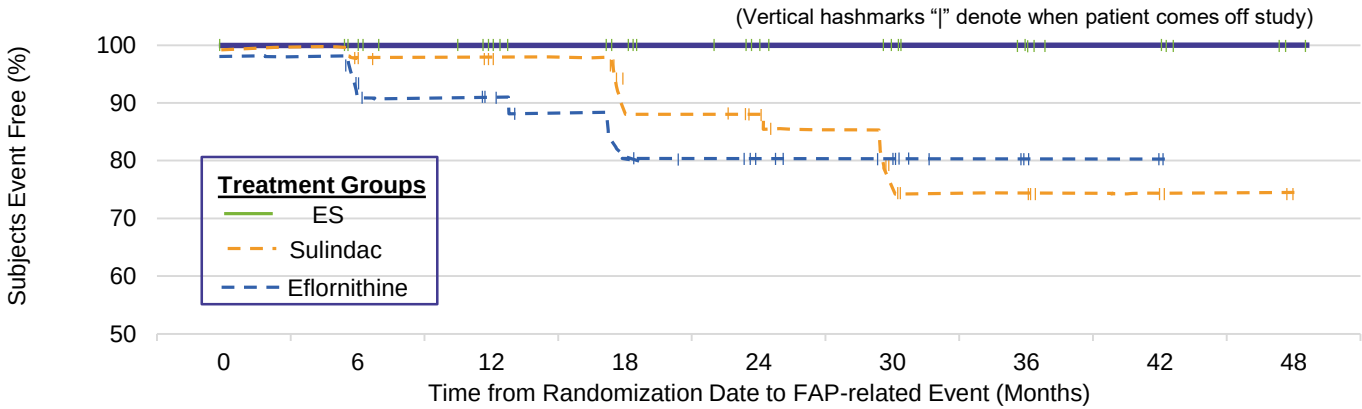
Surgery Events	ES Combo	Eflornithine	Sulindac	Overall
	(N=56)	(N=57)	(N=58)	(N=171)
Need colectomy	0	3	4	7
Need proctectomy	0	1	1	2
Need pouch resection	0	4	1	5
Total Surgical Events	**0**	**8**	**6**	**14**
Event Rate	**0/56 (0%)**	**8/57 (14%)**	**6/58 (10%)**	**14/171 (8%)**

No surgeries in Lower GI in ES Combo arm

Balaguer et al 2021 DCR
Note: Does not include adenoma removal

Flynpovi Combination Delays Need for Lower GI Surgery

Delay in Lower GI Surgery Events



Pts. on study									
ES	54	46	39	33	27	22	15	8	1
Sulindac	53	44	35	28	23	15	11	6	1
Eflornithine	51	41	34	28	16	11	4	2	0

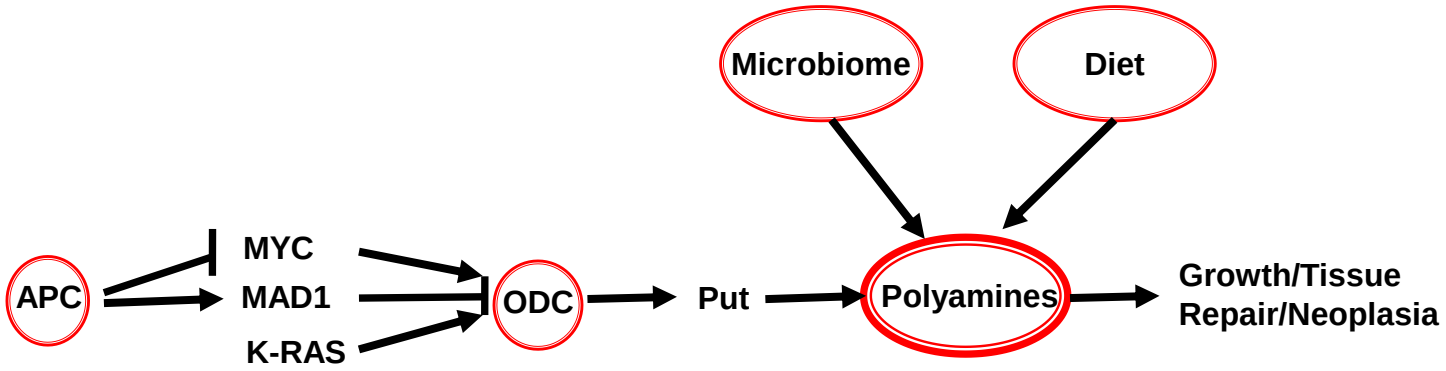
Hazard Ratio (95% CI) for having an Event
ES vs Sulindac = 0.000 (0.000, 0.483)
ES vs Eflornithine = 0.000 (0.000, 0.439)

Nominal P-Value
ES vs Sulindac = 0.0048
ES vs Eflornithine = 0.0031

In Subjects with Lower GI Anatomy (i.e., excludes 13 patients with ileostomy)

ES = eflornithine + sulindac combination or Flynpovi
Balaguer et al 2021 DCR

Rationale for Efficacy of Flynpovi in the Lower Gastrointestinal Tract of FAP Patients



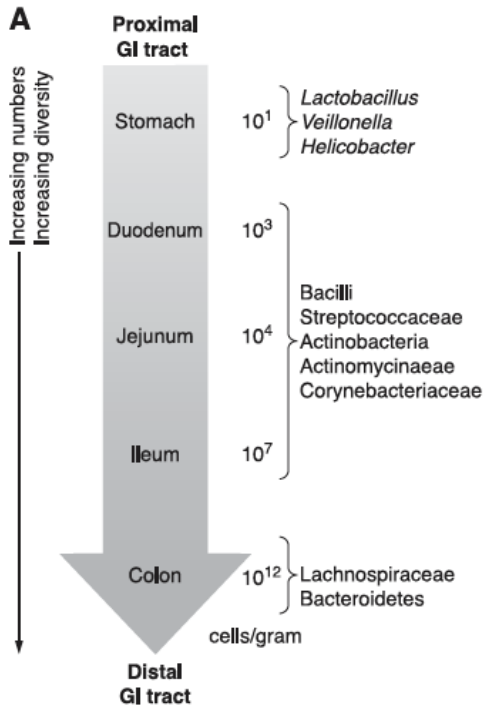
- Polyamines drive uncontrolled growth, polyposis, and cancer.
- Polyamines can by synthesized but also derived from the diet and microbiome.
- The mechanism of disease driven by polyamines which are upregulated in gastrointestinal mucosa in FAP patients - Eflornithine target, ODC is increased 2.5-fold with APC mutation
- The colon has 9 orders of magnitude more microbiome than upper GI that contribute to polyamines levels.

- **Flynpovi acts by a dual mechanism of action suppressing de novo synthesis and increasing the export of polyamines. The overall goal is to prevent or delay the onset of cancer through the regression or prevention of colonic adenomas through the reductions in polyamines.**

ODC = ornithine decarboxylase

High Concentrations of the Microbiota Contribute to the Impact of Polyamines in The LGI

- The microbiota increases steadily along the gastrointestinal tract.
  - **Small numbers in the stomach but very high concentrations in the colon.**
  - **Areas of the LGI contain up to <u>nine orders of magnitude (10^9)</u> more bacteria than that in the duodenum and other areas of the UGI.**
- Microbiota produces **biofilms which increase polyamines leading to colonic epithelial cell proliferation and colorectal cancer development**
- Colonic mucosa of FAP patients can harbour biofilms containing tumorigenic bacteria
- **Microbiota provides a potential mechanism of action for the enhanced efficacy of Flynpovi in the LGI via polyamines.**



Johnson et al 2015; Dejea et al 2018; Sekirov et al 2010

Adverse Events of Special Interest

Adverse Events of Special Interest	Flynpovi (Eflornithine + Sulindac Combination) N=56 Subjects (%)	Sulindac N=57 Subjects (%)	Eflornithine N=56 Subjects (%)
Anemia	1 (1.8)	5 (8.8)	2 (3.6)
Myelosuppression	0	1 (1.8)	0
Thrombocytopenia	0	3 (5.3)	1 (1.8)
Cardiovascular/Thrombotic events	1 (1.8)	1 (1.8)	1 (1.8)
Hearing impairment/Tinnitus	5 (8.9)	8 (14.0)	2 (3.6)
Non-bleeding GI event	33 (58.9)	25 (43.9)	28 (50.0)
Bleeding GI event	17 (30.4)	17 (29.8)	10 (17.9)
Headache/Migraine/Tension Headache	8 (14.3)	13 (22.8)	7 (12.5)
Dizziness/Vertigo	4 (7.1)	4 (7.0)	7 (12.5)

- **Comparable safety** amongst all treatment arms

Basis for FDA Complete Response Letter

- **Clinical**
  - Single pivotal study efficacy was based on exploratory post-hoc analysis using a modified primary endpoint in a subgroup of the intent-to treat population
    - Asked to perform a new study focused on FAP patients with an intact lower-GI tract which showed statistically significant effect in post-hoc analysis of FAP-310 trial
- **Quality**
  - Proposed dissolution condition for testing of Eflornithine is different from the FDA Guidance for Industry: Dissolution Testing and Acceptance Criteria for Immediate-Release Solid Oral Dosage Form Drug Products
    - ✓Revised dissolution specifications
  - Limited discriminating ability of the dissolution method for compression force and no discriminating ability for the magnesium stearate or sulindac particle size distribution
    - Dissolution method being revised to address concerns

Moving Ahead: New Phase III Trial

- **North America Licensee funding next trial**
- **FDA and EMA focus on new Phase III trial:**
  - Lower GI focus (LGI), informed by positive data from FAP-310 and regulatory feedback
  - Reaffirm current clarity & acceptable aspects of CMC, clin pharm, and non-clinical package
  - Initiate new LGI focused pivotal trial
- **FDA:**
  - Meeting to ensure alignment for new trial (via Licensee): completed 2H22
  - Initiate new trial: 2H23
- **EMA:**
  - Select National Competent Authority (NCA) engagement on new trial and clear registration path (Sweden/Netherlands based on CHMP feedback)
  - Follow up SAWP engagement with revised approached, informed by CHMP and NCA interactions

Summary of Approach to Approval

Strong Results in the Lower GI

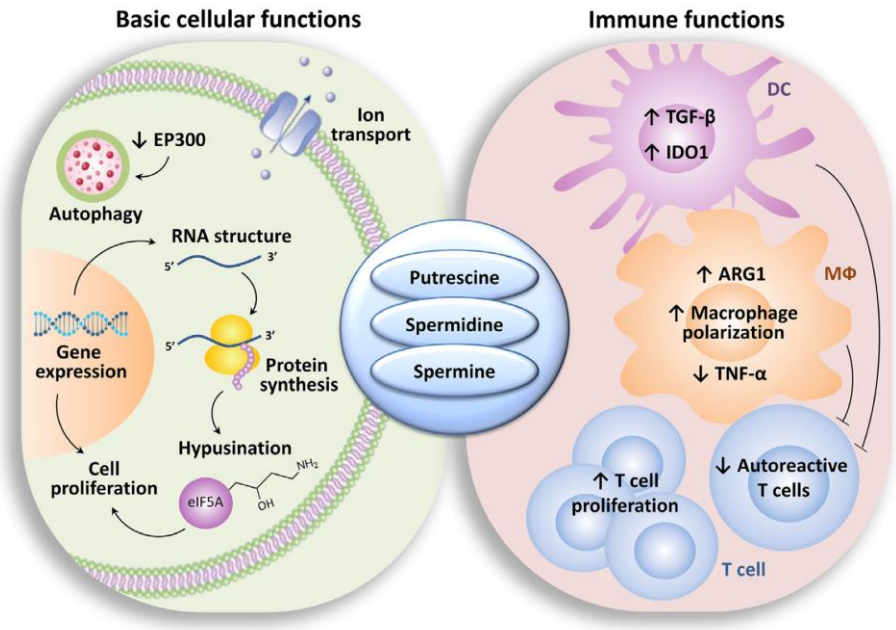
- ✓ Focus on Lower GI: statistically significant p-values and hazard ratios for clinically meaningful surgical endpoints—emphasized by regulators—in Phase III pivotal trial
- ✓ Expected safety profile over 2-4 years of daily dosing in Phase III pivotal trial
- ✓ Totality of the evidence includes mechanistic, preclinical, and clinical supportive data

No Other Approved Therapy for this Orphan Disease

Polyamines & Immune Dysregulation

Role of Polyamines in Immune Dysregulation

- Immune systems require multiple soluble and cellular components, including polyamines, for a normal immune function
- High levels of polyamines are present in tumor cells and in autoreactive B- and T-cells in autoimmune diseases
- Dysregulation of polyamines can result in:
  - Tumor immune evasion
  - Elevated cell stress
  - Increased autoimmunity
- Panbela's pipeline focuses on **resetting the polyamine pathway to restore normal immune function**



Proietti E et al 2020 Trends in Immunol

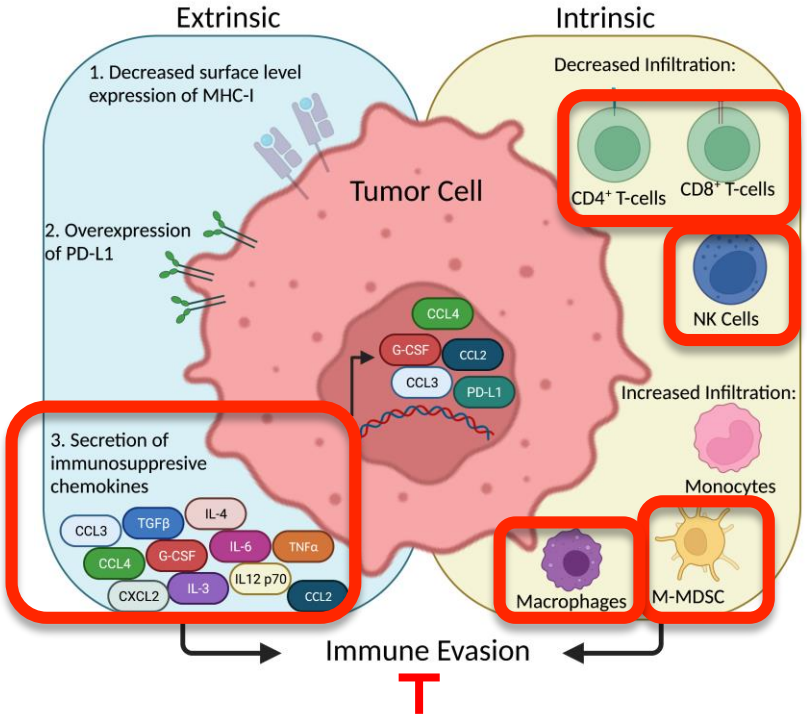
Role of Polyamines in Diffuse Large B Cell Lymphoma (DLBCL)

- MYC overexpression in relapsed/refractory DLBCL prior to CAR-T cell therapy negatively associated with durable response to CAR-T cell therapy.
- Polyamine metabolism upregulation through oncogenic MYC is a common metabolic irregularity in aggressive cancers, including lymphomas.
- Myeloid-derived suppressor cells (MDSCs) and higher IL-6 cytokine levels associated with reduced CAR-T cell expansion/poor anti-lymphoma responses after anti-CD19 CAR-T cell therapy.
- Polyamines have been reported to increase MDSCs and lead to an immunosuppresive tumor microenvironment.
- Polyamines have been shown to be important for effector function of several immune cell types including T cells.
- Circulating acetylated polyamine levels may function as a predictor of therapeutic outcome to CAR-T cell therapy.
- This suggests a possible strategy to target polyamine metabolism to augment the efficacy of CAR-T cell therapy.

Fahrmann JF et al 2022 Cell Reports Medicine

Rationale for Eflornithine/anti-PD-1 Combination

- Targeting Ornithine decarboxylase (ODC) using eflornithine unmasks antitumor T cell immune response by weakening of tumor-induced immune suppression that is mediated by Myeloid-Derived Tumor Suppressor Cells (MDSCs).
- The addition of anti-PD-1 prevents the functional exhaustion of these surviving T cells leading to enhanced antitumor effects.
- The combination of eflornithine/anti-PD1 prevents immune evasion
- **Targeting the polyamine pathway, treatment may reinvigorate the T cell–directed activity of an anti-PD-1 therapy and promote immune-mediated elimination of tumor cells.**



Modified from Tanaka M et al 2020 Cancers

CPP-1X-S

Rationale for CPP-1X-S (Eflornithine Sachets) in STK11 Mutant NSCLC

- In preclinical tumor models, eflornithine treatment improves anti-PD-1 efficacy by:
  - Increasing tumor-specific cytotoxic T-cell populations
  - Increasing expression of PD-1 on tumor-associated CD8+ T cells

- ~10% of NSCLC cancers have STK11 mutations
- STK11 mutant NSCLC tumors have:
  - Reduced cytotoxic T-cells infiltrates
  - Upregulation of the arginine pathway which utilizes ODC to increase levels of the polyamine putrescine
  - Respond poorly to immune checkpoint inhibitor therapy

Cancer Genomic Atlas Research 2014 Nature
Skoulidis F et al 2018 Cancer Discov
Skoulidis F et al 2019 JCO
Dryia P et al 2021 J Immunother
Alexander Et et al 2020 Mol Cancer Ther

STK11 Mutant NSCLC Investigator-Initiated Trial

*Collaborating with investigators at Moffitt Cancer Center on Phase I/II trial: "**Targeting Ornithine Decarboxylase** as an Immunotherapeutic Target in **STK11 (LKB1) Pathway-Deficient Non-Small Cell Lung Cancer**"*

Patient Population

- Stage IV NSCLC
- Immune Checkpoint Inhibitor Naïve
- STK11 mutant

Phase I
Standard Pembro + CPP-1X-S Dose Escalation (PD-L1≥1%)

Primary Endpoint: Toxicity

Phase II
Standard Pembro + CPP-1X-S (Dose from Phase I) (PD-L1≥1%)

Primary Endpoint: Efficacy (Response Rate)
Secondary Endpoints: Overall Survival, Progression-free Survival

Currently finalizing **trial protocol**, manufacturing **CPP-1X-S** (eflornithine sachet product), and completing **regulatory requirements**

PD-L1-programmed death ligand-1

Business Overview

Barriers to Entry – Flynpovi/CPP-1X

- **Orphan status (7 years exclusivity US; 10 years exclusivity EU) for CPP-1X**
  - Granted in US and EU for FAP
  - Granted in US and EU for Neuroblastoma
  - Granted in US for Gastric cancer and Pancreatic cancer
- **Pharmaceutical Composition Patent**
  - Fixed dose combination eflornithine + sulindac
  - Broadly nationalized with potential protection through 2037
- **Method of Use Patents/Patent Applications**
  - Fixed dose combination of eflornithine + sulindac and eflornithine single agent
  - “Theranostic” patents will afford protection to 2034 (“theranostic”= drug + diagnostic to guide therapy)
  - Use in Neuroblastoma protection through 2035
  - Use for Treating FAP with potential protection through 2040
  - Use in Treating Recent Onset Type 1 Diabetes with potential protection through 2041
  - New patents under consideration

Barriers to Entry – SBP-101

- **Orphan status (7 for years exclusivity US; 10 years exclusivity EU) for SPB-101**
  - Granted in US and EU for pancreatic cancer

- **METHODS FOR PRODUCING (6S,15S)-3,8,13,18-TETRAAZAICOSANE-6,15-DIOL**
  - PCT application filed January 29, 2019
  - Granted in United States, India
  - Pending in United States (CON), Europe, Australia, Canada, China and Japan
  - 20-year expiration date is January 29, 2039

- **DOSING REGIMENS AND METHODS FOR TREATING CANCER**
  - PCT application filed January 20, 2021
  - Pending in United States, Australia, Canada and Japan
  - Filing in progress in Europe (due August 20, 2022)
  - 20-year expiration date is January 20, 2041

Summary of Milestones

1H 2023

- Open Phase I Ovarian Cancer Trial
- Open- Phase I NSCLC Trial
- Open- Neoadjuvant Pancreatic Cancer Trial
- Open – Phase II Type I onset Diabetes Trial
- **Futility Analysis Phase III Colon Cancer Risk Reduction Trial (PACES)**
- **Publication of Phase I Type I early onset Diabetes Data**
- **Publication of Phase I Metastatic Pancreatic Trial Data**
- **Final Data – Phase I 1L mPC**
- **Gastric Cancer Prevention Phase II Results**

2H 2023

- Open FAP Registration Trial (funded by Licensee)
- **Phase I NSCLC Data**
- Open Phase II NSCLC Trial

1H 2024

- **Overall Survival Interim Analysis Phase III ASPIRE Trial**

ASCO-American Society of Clinical Oncology
GI-Gastrointestinal
mPC-Metastatic Pancreatic Cancer
FPI-First Patient In
FAP-Familial Adenomatous Polyposis
NSCLC – Non Small Cell Lung Cancer

Financial Position and Capital (unaudited)

Panbela	
Cash at 9/30/22	941,000
Estimated net proceeds from public sale of equity on 10/3/22	5,200,000
Estimated net proceeds sale of equity via Company ATM[(1)]	639,000
Proforma Cash Balance[(2)]	**6,780,000**
Debt 9/30/22	
Current (plus accrued interest)	**1,800,000**
Net of current portion	**5,194,000**
Common Stock at 9/30/22[(3)]	519,750
Shares issued on 10/3/22 and Prefunded warrants exercised prior to 12/31/22	502,500
Shares sold via Company ATM through 1/13/22[(1)]	154,909
Total Proforma Common Outstanding	**1,177,159**
Shares reserved for Options (WAE = $145.46)	100,578
Shares reserved for Warrants (WAE = 38.05)	889,911
Total Outstanding and Reserved	**2,167,648**

(1) Sales of equity and estimated net proceeds through 1/12/22 disclosed in 8-k dated 1/18/22
(2) Proforma Cash consists of 9/30/22 balance plus disclosed equity transactions subsequent to 9/30/22
(3) All Common stock, Option and Warrant amounts have been restated for 1 for 40 reverse stock split effected on 1/13/2023

Summary: Combination of PBLA and CPP

Unique Scientific Approach: Resetting dysregulation via polyamine pathway

- ✓ Broad, late-stage de-risked pipeline opportunities
- ✓ Multiple partnerships supporting development programs
- ✓ Significant Market Potential
- ✓ Multiple data readouts expected
- ✓ Experienced Management Team